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EXAMINER	Sharon L. Hurt
GROUP	1648
APPLICANT	Steven Jones et al.
SERIAL NO:	10/522,134
FILED	August 29, 2005
FOR	RECOMBINANT VESICULAR STOMATIS VIRUS VACCINES
FOR	
	VIRAL HEMORRHAGIC FEVERS

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Dear Sir:

DECLARATION

We, Steven Jones and Ute Stroeher of (National Microbiology Laboratory, 1015 Arlington Street, Winnipeg, MB R3E3R2, Canada) respectively do hereby solemnly swear that:

1. I, Steven Jones am a Head of Immunopathology, Special Pathogens Program

2. I, Ute Stroeher am a Head, Molecular Virology & Antiviral Approaches Unit

3. Regarding Ito, we note that this reference states 'because expression of Ebola virus GP on the cell surface does not induce polykaryon formation, regardless of the pH to which the GP is exposed (26), we could not

use this or similar assays to identify the fusion domain of the Ebola virus GP. Thus, we introduced amino acid substitutions into the putative fusion domain of the Ebola virus GP and examined the effect of these substitutions on the infectivity of VSVΔG* complemented with a GP mutant. The results suggest that the amino acids at position 524 to 539 do, in fact, constitute the fusion domain of the Ebola virus GP.' Page 8907, column 2, last paragraph.

Thus, Ito teaches using a recombinant form of VSV that contains the green fluorescent protein gene instead of the G protein gene and supplying a series of Ebola GP mutants *in trans* to determine what effect this had on virus entry.

That is, Ito does not teach inserting a VHF glycoprotein into the VSV so that the VHF glycoprotein replaces VSV G; rather, Ito teaches inserting fluorescent green protein into the genome in place of VSV G and supplying Ebola GP mutants in excess driven by a separate (extra-genomic) expression plasmid *in trans*. These particles, once created *in vitro*, can enter target cells only once and subsequently express green fluorescent protein but there is no subsequent production of recombinant virus particles because there is no glycoprotein expressed in these cells.

Thus, Ito teaches a particle that is not live or replication-competent or infectious but a gene transfer system using the Ebola GP to target GFP and consequently these particles are limited to one round of gene delivery.

Regarding the section on page 8908, 2nd column, this section refers to results shown in Figure 3 and the description of Figure 3 states 'VSVΔG*

complemented with Ebola virus GP and its mutants prepared as described previously (26)...' (page 8910, legend for Figure 3). Thus, in these cases, Ito does not teach 'a recombinant VSV expressing Ebola glycoprotein wherein the mutation reduced the infectivity of the VSVΔG by the incorporation of the Ebola virus glycoprotein into recombinant VSV particles'; rather, Ito teaches that with only a few exceptions, when mutant Ebola GP was supplied *in trans* with VSVΔG*, the mutant Ebola GPs were incorporated into the VSVΔG* particles equivalently to the wild type Ebola GP when supplied *in trans*. Thus, these particles are not expressing wild type Ebola GP or mutant Ebola GP – the Ebola GP is being supplied *in trans*. As such, these particles are not infectious and not replication competent as discussed above.

4. Regarding Khan, we note that Kahn teaches that in order to recover the VSVΔG-RSV G and VSVΔG-RSV F particles, they must be propagated on a modified BHK cell line which expresses VSV G (Kahn, page 11081, 2nd column, 1st complete paragraph). Thus, the particles taught by Kahn contain VSV G in addition to RSV F or RSV G. Neither RSV F nor RSV G are able to functionally replace VSV G as the dependency on VSV G clearly demonstrates. The presence of the VSV G in the particle limits the utility of the system because, if an immune response is elicited by the particle, there will also be neutralizing immunity against VSV G, meaning that the system can only be used in a single use. Specifically, subsequent use of the system will fail in a previously immunized individual because of the previous immune response against the VSV G (due to the previous exposure), and there will be no or only

limited immune response to the foreign protein on the surface of the particle. There is also the risk that the individual will have pre-existing neutralizing immunity against VSV G from a previous, non-vaccine exposure. Furthermore, the particle taught by Kahn does not meet the limitation in the claims that only the VHF glycoprotein is expressed on the surface of the particle.

Yet further, Kahn in fact discovered that VSVΔG-RSV G did not induce an immune response or protective immunity although VSVΔG-RSV F did. Specifically, Kahn states:

'VSVΔG-RSV G and VSVΔG-RSV F failed to induce serum RSV neutralizing titers as determined by the plaque reduction assay' (page 11083, column 1, 2nd complete paragraph).

'VSVΔG-RSV F, but not VSVΔG-RSV G, induced an RSV-specific antibody response (Fig. 4B). These results were in contrast to the neutralizing antibody titer results (Table 1), in which serum from mice immunized with VSVΔG-RSV F recombinants expressing RSV proteins failed to neutralize RSV.' (Kahn, page 11083, paragraph spanning columns 1 and 2).

'RSV replicated to significant titers in mice which were previously immunized with either wild-type VSV or VSVΔG (Fig. 5)... RSV was not detected in either BAL fluid or lung tissue from any mouse immunized with VSV-RSV G, VSV-RSV F, or VSVΔG-RSV F. VSVΔG-RSV G failed to protect from RSV replication.' (Kahn, page 11083, 1st complete paragraph).

Thus, Kahn teaches that VSVΔG-RSV G cannot elicit a protective immune response but that VSV-RSV G can. It is further of note that as discussed

above even the VSVΔG-RSV G contains VSV G supplied *in trans*. As such, these particles would have included VSV G and therefore would have been of limited utility as they could only be used once.

5. Thus, Ito teaches only that artificial over expression of Ebola GP supplied *in trans* from an extra-genomic expression plasmid *in vitro* can drive the formation of pseudotyped non-replicating gene delivery particles capable of delivering the GFP gene to target cells. Thus, Ito developed an *in vitro* system for studying target cell and receptor preferences for Ebola GP not a live replicating vaccine delivery system. Kahn teaches that VSVΔG-RSV G is not capable of inducing protective immune responses and that whilst VSVΔG-RSV F does induce neutralizing antibody responses, VSV G must be supplied *in trans* when the non-replicating psuedotyped particles are created for this system to be functional. Therefore, based on these references one of skill in the art would conclude that VSV G is required whether supplied *in cis* or *in trans* if VSV is to be used as a vaccine system and that only supplying VSV G *in cis* will result in replicating viral vaccine vectors. One of skill in the art would also conclude that supplying VSV G *in trans* reduces the system to a gene delivery system similar to replication deficient Adenoviruses, that is, a single use, non-replicating system.

6. In contrast, our goal was to generate replication competent virus particles which only display one glycoprotein (the foreign glycoprotein) on the virion surface. In this case, the immune response would be directed only against the foreign glycoprotein and these virus particles would be replication competent in a host where VSV G is not be provided *in trans*, but must, if

required, be there *in cis*. The inclusion of VSV-G in any vaccine vector is clearly undesirable, for two reasons: it has been demonstrated that the VSV G is a pathogenicity factor, causing neurovirulence, and it is a target for neutralizing immunity that limits vaccine efficacy. By replacing the native glycoprotein with a foreign one, we have ablated the neurovirulence and ensured that any pre-existing neutralizing immunity to VSV G will not impact vaccine efficacy. Furthermore, we have ensured that by replacing functional glycoproteins, we can use the same core vector system to deliver VHF vaccines serially i.e. Ebola followed by Marburg followed by Lassa and that the existing immunity to the previously delivered glycoproteins will not impact later immunizations.

7. It was considered by those with skill in the art that VSV particles exclusively expressing and encoding a foreign glycoprotein completely devoid of VSV G would not be capable of assembling correctly in the host cell resulting in the production of defective and non-replicating viral particles that would not have been useful as vaccine vectors or even as research tools. The concern was mainly that the foreign glycoprotein could not be incorporated efficiently and because of this, infectious VSV particles could not be generated.

The prior art has shown that this concern was legitimate as multiple research articles demonstrate the dependency on VSV G provided either *in trans* or *in cis*. (Kahn JS, Roberts A, Weibel C, Buonocore L, Rose JK. Replication-competent or attenuated, nonpropagating vesicular stomatitis viruses expressing respiratory syncytial virus (RSV) antigens protect mice against RSV challenge. J Virol. 2001 Nov;75(22):11079-87; Kretzschmar E, Buonocore L,

Schnell MJ, Rose JK. High-efficiency incorporation of functional influenza virus glycoproteins into recombinant vesicular stomatitis viruses. J Virol. 1997 Aug;71(8):5982-9; Rose NF, Roberts A, Buonocore L, Rose JK. Glycoprotein exchange vectors based on vesicular stomatitis virus allow effective boosting and generation of neutralizing antibodies to a primary isolate of human immunodeficiency virus type 1. J Virol. 2000 Dec;74(23):10903-10; Roberts A, Kretzschmar E, Perkins AS, Forman J, Price R, Buonocore L, Kawaoka Y, Rose JK. Vaccination with a recombinant vesicular stomatitis virus expressing an influenza virus hemagglutinin provides complete protection from influenza virus challenge. J Virol. 1998 Jun;72(6):4704-11). The latter two authors also discuss how the inclusion of VSV-G in the vaccine limits the potential for multiple doses and Rose 2000 actually constructed multiple VSV vectors encoding different serotypes of VSV G to overcome this issue and this was required specifically because VSV-G was considered essential for the success of the vaccine system.

Thus, the balance of this evidence made it appear extremely unlikely that we would be successful in rescuing the VSV delta G ZEBOV GP. Furthermore paper by Yang (Yang ZY, Duckers HJ, Sullivan NJ, Sanchez A, Nabel EG, Nabel GJ. Identification of the Ebola virus glycoprotein as the main viral determinant of vascular cell cytotoxicity and injury. Nat Med. 2000 Aug;6(8):886-9) indicated if we were successful, the resulting virus would be highly pathogenic chimera as it included the primary virulence factor for Ebola virus, the glycoprotein. Consequently, we were required to work with these recombinant viruses in the BSL-4 laboratory. This is the highest level of biological

containment available. Workers in BSL 4 facilities use total encapsulation suits with breathing air supplied from the outside. This level of containment is reserved for the most dangerous pathogens in the world, such as Ebola, Marburg, Lassa fever virus and Smallpox. The fact that we were required to work with the VSV recombinant vectors under these conditions is a clear indication of the concern that these vectors caused in the community of experts.

This clearly establishes that our findings that (a) the Ebola virus glycoprotein can functionally replace the authentic VSV glycoprotein rendering a fully replication competent virus and (b) that this virus would in fact be non pathogenic in all animal models tested as well as attenuated in tissue culture could not have been anticipated by one of ordinary skill of the art and therefore constituted a significant discovery.

8. Specifically, it was reported by Yang et al., 2000 that the GP of Ebola was the main viral determinant of vascular cell toxicity and vascular injury and directly contributed hemorrhage during infection. Furthermore, it was known that human vasculature was only adversely effected by the Ebola glycoproteins from viruses known to be virulent to humans and that those Ebola glycoproteins virulent in monkeys but not humans caused no damage to human vasculature. Thus, the inclusion of the Ebola GP meant that the recombinant virus carried the most important virulence determinant and therefore disease symptoms were anticipated. Concern was significant enough that at this time the US-CDC added the Ebola glycoprotein to the select agent list to ensure the safety of Americans from this potentially harmful protein. Accordingly, providing

a live, propagating, replication competent virus particle encoding an Ebola or Marburg or other VHF glycoprotein was considered to be risky and dangerous requiring the maximum level of containment. However, we were surprised to find that there were no associated side effects. As such, while supplying *in cis* rather than *in trans* may have been considered easier, it was taught against by the prior art and therefore was not done by others out of concern for potential side effects as well as being considered unlikely to produce a functioning virus.

The particle we developed was derived from a DNA copy of the VSV genome completely lacking the native VSV Glycoprotein. As discussed at length above, it is impossible for the proteins encoded for by this version of the genome to form infectious particles due to the complete deletion of the glycoprotein gene. Therefore, we inserted the glycoprotein gene from Ebola virus into the VSV genome, thereby functionally replacing the native glycoprotein. Surprisingly, these particles were found to assemble properly and bud from the cell surface. They contained only the Ebola GP (or other donor GP such as Lassa or Marburg) and were capable of productively infecting target cells *in vitro*, i.e. when added to fresh uninfected cells, they underwent multiple rounds of replication, exponentially increasing in number over a few days. This teaches that, contrary to the then widely accepted truth, it was possible to functionally replace the glycoprotein of VSV with another glycoprotein from a completely different virus genus. When these viral particles were injected into mice, they caused no pathology, indicating that, contrary to that taught by Yang et al., the glycoprotein was not the major virulence determinate even when expressed in an

infectious viral system. These particles were thus live-replicating recombinant viruses capable of growing in target cells and amplifying virus particles, in contrast to the gene delivery systems taught by Kahn or Ito (delta VSV G in combination with not functional foreign glycoprotein results in not replicating particles).

10. Contrary to the teachings of Vanderzanden, vaccination against Ebola using DNA encoding the Ebola virus GP only, whether delivered as naked DNA, by Vaccinia virus vector, by Venezuelan Equine Encephalomyelitis Virus replicon, or by Baculovirus, failed to protect 100% of rodents immunized and totally failed to protect non human primates. Sullivan (Sullivan NJ, Sanchez A, Rollin PE, Yang ZY, Nabel GJ. Development of a preventive vaccine for Ebola virus infection in primates. Nature. 2000 Nov 30;408(6812):605-9) taught that multiple doses of DNA encoding both GP and NP protected 8/8 guinea pigs and when adenovirus delivery was added as a final step to that schedule non-human primates were protected. This teaches that DNA immunization with the GP alone is unlikely to protect animal models and consequently humans from Ebola virus infection and that the nucleoprotein (the most abundant viral protein) is required for successful vaccination. The particles taught by Kahn and Ito are DNA delivery vehicles and thus on with skill in the art would believe based on this significant existing data that non replicating DNA vaccine vectors must include the genes for more than just the Ebola GP to be effective as well as an absolute requirement for the VSV G to be supplied *in cis* or *in trans*. The live replicating VSVΔG-ZEBOVGP particle is the only platform delivering only Ebola GP to

protect 100% of rodents and non-human primates. In addition, it was the first platform to be able to protect non-human primates following a single dose and the only platform capable of providing post exposure protection. These features are directly related to the live replicating nature of the vaccine vector. The single dose immunization works because the vaccine replicates following immunization and this amplifies the dose delivered. We believe that the post exposure immunization is possible because the vaccine is targeted by the Ebola GP on it's surface to the exact same cells that the Ebola virus is trying to infect. In contrast to the native Ebola virus, which suppresses antiviral responses in host cells and inhibits adaptive immune responses, the VSV vaccine stimulates both antiviral and adaptive immunity allowing the host to overcome the Ebola infection. Non-replicating DNA vectors and protein subunits cannot achieve this effect. This makes the VSV system unique and extremely powerful. Whilst post exposure vaccination has been shown to be possible for diseases such as rabies and smallpox where the incubation time is measured in weeks, no one could have predicted that the VSVΔG-ZEBOVGP could protect against Ebola infection as it is a highly acute disease particularly in the animal models tested with an incubation time of 2-3 days and a mean time to death of 6-8 days.

11. In summary, we have discovered that a VSVΔG particle can be constructed which has only a VHF glycoprotein and no VSV glycoprotein that can be used safely as a vaccine based on its ability to propagate. This is in contrast with the prior art which teaches that VSV G must be supplied in trans and even then an immune response may not be elicited (Kahn) and that a

propagating virus expressing one VHF glycoprotein (Zaire strain of Ebola) can cause symptoms associated with VHF and accordingly may not be safe. Ito teaches that Ebola GP can confer infectivity to a VSVΔG when supplied in trans which was done as a precaution against presumed harmful effects of the Ebola GP protein. Accordingly, the prior art taught that a VSVΔG-VHF G construct may not elicit an immune response even if VSV G was supplied in trans but such a construct could have considerable side effects. We found surprisingly that neither was the case and that a safe and effective vaccine could be developed.

We declare that all statements made herein of our own knowledge are true and that all statements made on information and belief are believed to be true; and further that these statements were made with the knowledge that willful false statements and the like so made are punishable by fine or imprisonment or both under Section 1001 of Title 18 of the United States Code and that such willfull false statements may jeopardize the velidity of the instant patent application or any patent issuing therefrom.

Steven Jones

Ute Stroeher

BIOGRAPHICAL SKETCH

Provide the following information for the key personnel in the order listed on Form Page 2
Photocopy this page or follow this format for each person.

NAME	POSITION TITLE
Dr. Steven M Jones	Head, Immunopathology, Emerging Bacterial Diseases and Microbiological Emergency Response Teams

EDUCATION/TRAINING *(Begin with baccalaureate or other initial professional education, such as nursing, and include postdoctoral training.)*

INSTITUTION AND LOCATION	DEGREE *(if applicable)*	YEAR(s)	FIELD OF STUDY
University of Plymouth	Ph. D.	1992-1996	Microbiology and Immunology
University of Plymouth	BSc(Hons) 1st Class	1989-1992	Biological Sciences Cell Biology and Microbiology

RESEARCH AND PROFESSIONAL EXPERIENCE: Concluding with present position, list, in chronological order, previous employment, experience, and honors. Include present membership on any Federal Government public advisory committee. List, in chronological order, the titles, all authors, and complete references to all publications during the past three years and to representative earlier publications pertinent to this application. If the list of publications in the last three years exceeds two pages, select the most pertinent publications. **DO NOT EXCEED TWO PAGES.**

1997-1999 **Post-Doctoral Scientist,** Plague Vaccine Research Program, Chemical and Biological Defense Establishment Porton Down SALISBURY, UK

1999-2000 **Senior Scientist** Glanders, Meliodoisis and Tularemia Vaccine programs Chemical and Biological Defense Establishment Porton Down SALISBURY, UK

2000-2001 **Scientific Leader Cellular Immunology** Group Plague Vaccine Clinical Trials and Glanders, Meliodoisis and Tularemia Vaccine programs Chemical and Biological Defense Establishment Porton Down SALISBURY, UK

2001- **Research Scientist RES-02 Head of Immunopathology Unit,** National Laboratory for Zoonotic Diseases and Special Pathogens, Winnipeg Canada

2002-2005 **Adjunct Professor of Immunology** University of Manitoba Medical School Department of Immunology

2005- **Assistant Professor of Immunology** University of Manitoba Medical School Department of Immunology

2005- **Adjunct Professor Medical Microbiology** University of Manitoba Medical School Department of Medical Microbiology

2007- **Research Scientist RES-05 Head of Immunopathology Unit,** National Laboratory for Zoonotic Diseases and Special Pathogens, Winnipeg Canada

Other experience/ Appointments

1999-2001 **Deputy Study Director** GLP plague vaccine trials. Chemical and Biological Defense Establishment Porton Down SALISBURY, UK

2002 **Independent Area Expert (AE)** for the Military Infectious Diseases Research Program Review (MIDRP). Hantavirus , Hemorrhagic Fever, Rift Valley Fever, and Lassa viruses. United States of America, Department of Defense.

2004- Chemical, Biological Radionuclear Research and Technology Initivative (CRTI) Bio-Cluster Committee member

2005 Chief, Marburg Virus Outbreak Field Laboratory, Uige ANGOLA,

2005- Ontario Provincial Smallpox Preparedness Committee

2006- Interpol External Expert Instructor, Regional Bioterrorism Workshops

2006- Chemical, Biological Radionuclear Research and Technology Initivative (CRTI) Chemical Cluster Committee Toxins Working Group Member

2006- Chemical, Biological Radionuclear Research and Technology Initivative (CRTI) Forensics Cluster Committee Member

2006- Member of the AUS/CAN/UK/US Technical Support Working Group for Chemical and Biological Defence

2007-	Member of the AOAC International International Methods Committee on Biological Threat Agents
2008-	Head of Delegation of the AUS/CAN/UK/US Technical Support Working Group for Chemical and Biological Defence
2008-	Member IAB for CIHR III.
2009-	Co-Chair WHO Emerging and Dangerous Pathogens Laboratory Network
2009-2010	Head of Bioterrorism Response for the Vancouver Olympics and Pararlympic Games

Honors:

2001-2004	**Research Fellowship Award:-** Dendritic cell vaccines for intracellular pathogens. United Kingdom Ministry of Defence.
2007	**Commanders Commendation** from The Commander Canadian Special Forces Command for Contributions to improving Canadian National Security through training provded to the Special Forces

Research Projects Ongoing or Completed During the Last 3 Years:

Federal and non-federal support). Begin with the projects that are most relevant to the research proposed in this application. Briefly indicate the overall goals of the projects and your role (e.g. PI, Co-Investigator, Consultant) in the research project. Do not list award amounts or percent effort in projects.

On Going

CRTI 08-0181TD Jones PI 2009-2013 **$ 3,000,000**
Detection and Identification Assay Validation Program for Biothreat Agents

CRTI-08-0203RTD Aomoko PI Jones Co-PI 2009-2012: **$2,250,358.00**
Science and Technology Solutions to Mitigate Vulnerabilities in Canada's Food Supply

CRTI 07-0219RD Jones PI 2008- 2008-2013 **$ 2,740,000**
Microbial Forensics

CRTI 05-78RTD JONES 2006-2013 **$ 1,852,048**
Development of live replicating viruses as vaccines and therapies for Viral Hemorrhagic fever viruses
Role PI

NIH NAIAD SARS grant Ultrasensitive diagnostics of SARS viruses and their antigens (UO1A1061233-01)
Suresh
$915,000
Role Co-PI

CRTI 02-006RTD CZUB **$ 1,058,313**
"Rapid Induction of Innate Immunity at Mucosal Surfaces" The project aims to use a number of approaches to develop CpG motifs to rapidly generate enhanced innate immune responses to Filovirus and *Yersinia pestis* infection. 2003-2006
Role Co-PI

Completed
Canadian Institute of Health Research (CIHR) Feldmann (PI) / 03/2005– 02/2005
Biosynthesis, targeting and function of hantaviral glycoproteins
RoleCo-PI

CRTI 02- 0087RTD JONES **$ 2,316,206**
"Development of Therapeutic Antibodies for Filovirus Infection" The project aims to use a number of approaches to develop an antibody therapy for Filovirus infection. 2003-2006
Role PI

Canadian Institute of Health Research (CIHR/MHRC) (MOP – 99822)
Feldmann (PI) / 03/2003 – 02/2005
Biosynthesis, targeting and function of hantaviral glycoproteins. Partnership Program (Canadian Institutes of Health Research (CIHR) and Manitoba Health Research Council (MHRC) (2003-2005)
RoleCollaborator

Generic Therapy against BW agents JonesUK Ministry of Defence 2001-2004 $ **1,540,939**
Role PI

Generic BW diagnostic systems (Mass Spectrometry) Jones
UK Ministry of Defence 2001-2004 $ **960,419**
Role PI

Publications:

Factors Associated with Marburg Hemorrhagic Fever: Analysis of Patient Data from Uige, Angola. Roddy P, Thomas SL, Jeffs B, Nascimento Folo P, Pablo Palma P, Moco Henrique B, Villa L, Damiao Machado FP, Bernal O, **Jones SM**, Strong JE, Feldmann H, Borchert M. J Infect Dis. 2010

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Qiu X, Fernando L, Alimonti JB, Melito PL, Feldmann F, Dick D, Ströher U, Feldmann H, **Jones SM**. Mucosal immunization of cynomolgus macaques with the VSVDeltaG/ZEBOVGP vaccine stimulates strong ebola GP-specific immune responses. PLoS ONE. 2009;4(5):e5547. Epub 2009 May 14.

Groseth A, Charton JE, Sauerborn M, Feldmann F, **Jones SM**, Hoenen T, Feldmann H. The Ebola virus ribonucleoprotein complex: A novel VP30-L interaction identified. Virus Res. 2008 Dec 15.

Geisbert TW, Daddario-Dicaprio KM, Lewis MG, Geisbert JB, Grolla A, Leung A, Paragas J, Matthias L, Smith MA, **Jones SM**, Hensley LE, Feldmann H, Jahrling PB. Vesicular stomatitis virus-based ebola vaccine is well-tolerated and protects immunocompromised nonhuman primates. PLoS Pathog. 2008 Nov;4(11):e1000225.

Geisbert TW, Daddario-Dicaprio KM, Geisbert JB, Reed DS, Feldmann F, Grolla A, Ströher U, Fritz EA, Hensley LE, **Jones SM**, Feldmann H Vesicular stomatitis virus-based vaccines protect nonhuman primates against aerosol challenge with Ebola and Marburg viruses. Vaccine. 2008 Dec 9;26(52):6894-900

Geisbert TW, Daddario-DiCaprio KM, Williams KJ, Geisbert JB, Leung A, Feldmann F, Hensley LE, Feldmann H, **Jones SM**. Recombinant vesicular stomatitis virus vector mediates postexposure protection against Sudan ebola hemorrhagic fever in nonhuman primates.J Virol. 2008 Jun;82(11):5664-8

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Shimojima M, Takada A, Ebihara H, Neumann G, Fujioka K, Irimura T, **Jones S**, Feldmann H, Kawaoka Y. Tyro3 family-mediated cell entry of Ebola and Marburg viruses. J Virol. 2006 Oct;80(20):10109-16

Melito PL, Qiu X, Fernando LM, deVarennes SL, Beniac DR, Booth TF, **Jones SM**. The creation of stable cell lines expressing Ebola virus glycoproteins and the matrix protein VP40 and generating Ebola virus-like particles utilizing an ecdysone inducible mammalian expression system.J Virol Methods. 2008 Mar;148(1-2):237-43.

Jones SM. , Ströher U., Fernando L., Qiu X.,, Alimont JB.,, Melito P., Bray M., Klenk H-D, Feldmann H. Assessment of a Vesicular Stomatitis Virus–Based Vaccine by Use of the Mouse Model of Ebola Virus Hemorrhagic Fever Journal of Infectious Diseases S2 Nov 2007:
196 E-publication ahead of print.

Ebihara H., Theriault S., Neumann G., Alimonti JB., Geisbert JB., Hensley LE., Groseth A., **Jones SM.**, Geisbert TW., Kawaoka Y., and Heinz Feldmann In Vitro and In Vivo Characterization of Recombinant Ebola Viruses Expressing Enhanced Green Fluorescent Protein Journal of Infectious Diseases S2 Nov2007:196 E-publication ahead of print.

Kingston R, Burke F, Robinson JH, Bedford PA, **Jones SM**, Knight SC, Williamson ED.The fraction 1 and V protein antigens of Yersinia pestis activate dendritic cells to induce primary T cell responses.Clin Exp Immunol. 2007 Sep;149(3):561-9

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Shahhosseini S, Das D, Qiu X, Feldmann H, **Jones SM**, Suresh MR. Production and characterization of monoclonal antibodies against different epitopes of Ebola virus antigens. J Virol Methods. 2007 Mar 17

Williamson ED, Stagg AJ, Eley SM, Taylor R, Green M, **Jones SM**, Titball RW. Kinetics of the immune response to the (F1+V) vaccine in models of bubonic and pneumonic plague. Vaccine. 2007 Jan 22;25(6):1142-8. Epub 2006 Oct

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*Joint first authorship

Books, book chapters and Review Articles

Lacquement, A., **Jones, S.,** Artsob, H., Feldmann, H. Bioterrorism and Infectious Agents: A New Dilemma for the 21st Century Hemorrhagic Fever Viruses as Biological Weapons. Book

Other Publications

1. Feldmann H., Wahl-Jensen V., **Jones SM.**, Ströher U. Ebola virus ecology: a continuing mystery Trends Microbiol 2004 Oct;12(10):433-7.

2. Berry JD, **Jones S**, Drebot MA, Andonov A, Sabara M, Yuan XY, Weingartl H, Fernando L, Marszal P, Gren J, Nicolas B, Andonova M, Ranada F, Gubbins MJ, Ball TB, Kitching P, Li Y, Kabani A, Plummer F. Development and characterisation of neutralising monoclonal antibody to the SARS-coronavirus. J Virol Methods. 2004;120(1):87-96.

3. Stroher U, DiCaro A, Li Y, Strong JE, Aoki F, Plummer F, **Jones SM**, Feldmann H. Severe acute respiratory syndrome-related coronavirus is inhibited by interferon- alpha. J Infect Dis. 2004;189(7):1164-7.

4. Tang P, Louie M, Richardson SE, Smieja M, Simor AE, Jamieson F, Fearon M, Poutanen SM, Mazzulli T, Tellier R, Mahony J, Loeb M, Petrich A, Chernesky M, McGeer A, Low DE, Phillips E, **Jones S**, Bastien N, Li Y, Dick D, Grolla A, Fernando L, Booth TF, Henry B, Rachlis AR, Matukas LM, Rose DB, Lovinsky R, Walmsley S, Gold WL, Krajden S; Ontario Laboratory Working Group for the Rapid Diagnosis of Emerging Infections. Interpretation of diagnostic laboratory tests for severe acute respiratory syndrome: the Toronto experience. CMAJ. 2004;170(1):47-54.

5. Krokhin O, Li Y, Andonov A, Feldmann H, Flick R, **Jones S**, Stroeher U, Bastien N, Dasuri KV, Cheng K, Simonsen JN, Perreault H, Wilkins J, Ens W, Plummer F, Standing KG. Mass Spectrometric Characterization of Proteins from the SARS Virus: A Preliminary Report.. Mol Cell Proteomics. 2003;2(5):346-56. Epub 2003 May 29.

6. Marra MA, Jones SJ, Astell CR, Holt RA, Brooks-Wilson A, Butterfield YS, Khattra J, Asano JK, Barber SA, Chan SY, Cloutier A, Coughlin SM, Freeman D, Girn N, Griffith OL, Leach SR, Mayo M, McDonald H,

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7. **Jones SM**, Ellis JF, Russell P, Griffin KF, Oyston PC. Passive protection against Burkholderia pseudomallei infection in mice by monoclonal antibodies against capsular polysaccharide, lipopolysaccharide or proteins. J Med Microbiol. 2002;51(12):1055-62.

8. **Jones SM**, Day F, Stagg AJ, Williamson ED. Protection conferred by a fully recombinant sub-unit vaccine against Yersinia pestis in male and female mice of four inbred strains. Vaccine. 2000;19(2-3):358-66.

9. Bennett AM, Elvin SJ, Wright AJ, **Jones SM**, Phillpotts RJ. An immunological profile of Balb/c mice protected from airborne challenge following vaccination with a live attenuated Venezuelan equine encephalitis virus vaccine. Vaccine. 2000;19(2-3):337-47.

10. Eyles JE, Williamson ED, Spiers ID, Stagg AJ, **Jones SM**, Alpar HO. Generation of protective immune responses to plague by mucosal administration of microsphere coencapsulated recombinant subunits. J Control Release. 2000 ;63(1-2):191-200.

11. Miller J, Williamson ED, Lakey JH, Pearce MJ, **Jones SM**, Titball RW. Macromolecular organisation of recombinant Yersinia pestis F1 antigen and the effect of structure on immunogenicity. FEMS Immunol Med Microbiol. 1998 ;21(3):213-21.

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PERSONAL DATA

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UNIVERSITY EDUCATION

1990-1995	Biological Studies (2 semesters) & Human Biology (8 semesters), Philipps-University of Marburg, Germany.
1995-1996	Diploma, Institute for Virology, Philipps-University of Marburg, Germany.
	Major subject: Virology; minor subjects: Immunology & Molecular Biology
	Thesis: 'Identification and characterization of a nonstructural protein of Marburg virus'.
1997-2001	Ph.D. in Virology (Dr. rer. physiol.), Institute for Virology, Philipps-University of Marburg, Germany.
	Dissertation: 'Filovirus-induced Activation of human Endothelial cells and Monocytes/ Macrophages'.
	Date of defense: October 2004

POSITIONS AND EMPLOYMENT

1997 – 2001	Research Assistant, Institute of Virology, University of Marburg, Germany
2001 – 2004	Postdoctoral fellow, Special Pathogens Program, NML, PHAC, Winnipeg, Canada
2004 – 2006	Research Associate, Special Pathogens Program, NML, PHAC, Winnipeg, Canada
Since 2007	Research Scientist, Head of Molecular Virology & Antiviral Approaches, Special Pathogens Program, NML, PHAC, Winnipeg, Canada
Since 2008	Adjunct Professor, Department of Medical Microbiology, University of Manitoba
2009	Acting Chief Special Pathogens Program NML, PHAC (1 Feb – 30 Apr)

PROFESSIONAL ACTIVITIES & TRAINING

2005:	WHO consultant – Marburg virus outbreak in Uige, Angola. Established new molecular assays for diagnosis in the field and tested samples
2006:	Special Adviser (Laboratory Specialist) for WHO in Tajikistan (WHO mission to assess and strengthen national Avian Influenza preparedness. Provided assistance to the Ministry of Health and the Ministry of Agriculture of Tajikistan to identify a national reference laboratory. I was responsible to review and assess the preparedness of the country in respect to Avian Influenza diagnostic, including specimen collection, shipment, laboratory capacity, biosafety, and testing algorithm.
2007:	Essentials of Managing in the Public Service for new Executives. (Canada School for Public Service, Ottawa)
2007:	Infectious Disease Epidemiology; EPIET (European Programme for Intervention Epidemiology Training) Introductory Course, 2007; Lazareto, Menorca.
2009:	Mexico City, provided technical support to establish molecular diagnostic for swlH1N1 and provided training.
2009:	Incident Command System course (ICS 100) Emergency Operations Centre Level I & Level II (EM 7110 Emergency Operations Centre Level III (Operations & Logistics & Planning) (provided by Justice Institute of British Columbia, Emergency Management Division)
2009:	MERT training with Royal Canadian Mounted Police (Prince Edward Island) (lab manager of mobile laboratory, chain of custody, etc)
2009:	Team lead, MERT deployment to Trinidad & Tobago during Commonwealth Head of Governments meeting. Provided support to CAREC (The Caribbean Epidemiology Centre) for potential warfare agents (bacterial/viral) detection.
2010:	Team lead, MERT deployment to Whistler during Winter Olympic Games.

REVIEWER OF SCIENTIFIC JOURNALS

Archives of Virology, Biochemistry Insights, Future Medicine, Journal of General Virology, Molecular Aspects of Medicine, Virology Journal

RESEARCH INTERESTS

ANTIVIRALS

My main research interests are the identification of potential cellular and viral targets for the development of antiviral drugs for BSL-4 viruses, the development of *in vitro* screening assays for antiviral compounds, and understanding the mode of action of effective compounds. As viral targets I am particularly interested in viral glycoproteins and their proteolytical processing by cellular proteases. Other viral targets we are investigating are the matrix proteins of filoviruses and arenaviruses. By understanding their role in the assembly process of virus morphogenesis we are hoping to find ways to interfere with this crucial step in virus replication.
In addition, we are looking at cellular protein kinases, which are known to become activated in infection, and their role in the virus life cycle as potential targets for antiviral therapy.
An important goal for the future is the development of screening tools for BSL-4 viruses in high containment. So far we genetically engineered a *Zaire ebolavirus* encoding the green fluorescent protein, which therefore serves as an ideal tool to screen antiviral compounds in less time than classical approaches in BSL-4.

REVERSE GENETICS

To elucidate functional aspects of viral glycoproteins I used reverse genetics to create recombinant vesicular stomatitis viruses (VSV), which express the glycoproteins of the Marburg, Ebola, Lassa, Junin and Machupo viruses. Their advantages over wild-type lie in a much shorter replication time and the significantly lower level of bio-containment required for their use. This work was started in Marburg, Germany and has been successfully continued in our program in Winnipeg. They are meanwhile used for various purposes *in vitro* and *in vivo* and has led to several high quality publications:

- As screening tools for antiviral drugs targeting the glycoprotein
- Tropism studies
- Epitope mapping
- Vaccine candidates (Ebola, Marburg, Lassa, Machupo, Junin)

Especially for antiviral screening purposes we generated recombinant VSV encoding the green fluorescent protein in addition to the foreign glycoproteins of the New World arenaviruses Junin, Machupo, Sabia, Guanarito and Chapare virus.

We generated recombinant rabies viruses encoding glycoproteins of Marburg and Ebola virus. Their potential as a vaccine platform was evaluated in comparison to the existing VSV platform. Preliminary data show that the rabies platform doesn't protect mice against lethal challenge with mouse-adapted *Zaire ebolavirus*.

STRUCTURE-FUNCTION

The RNA dependant RNA polymerase (RdRp) of RNA viruses is a prime target for the development of antivirals. So far structural data for the RdRp of negative-sense single stranded viruses don't exist.
In order to study the structure of the Marburg virus RdRp and verify postulated domains, we are using structural biology, mutational analysis and reverse genetics systems developed for Marburg virus. This work is done in collaboration with the Department of Chemistry, University of Manitoba, Winnipeg.
(NSERC post-doc)

DIAGNOSTIC

I have established and improved RT-PCR assays (real time and/or standard) for viruses of the families *Arenaviridae* (Lassa virus, Machupo virus, Junin virus), *Filoviridae* (Marburg virus, Ebola virus), *Coronaviridae* (SARS), *Flaviviridae* (West Nile virus), *Rhabdoviridae* (Vesicular Stomatitis virus), *Orthomyxoviridae* (Influenza A virus), and *Bunyaviridae* (Rift Valley Fever Virus) which are used for diagnosis and/or research purposes.

I'm experienced in performing serological assays including IgG & IgM ELISA, immunoblot analysis, immunofluorescence analysis, and haemagglutination inhibition assays, as well as virus isolation (*in vitro*), haemagglutination tests, and neutralization tests.

As part of a collaboration with PAHO I am establishing ELISA assays (antigen production, generation of antibodies) for the diagnosis of South American Arenaviruses.

We developed antigen and antibodies for the diagnosis of Rift valley fever virus (CRTI project)

PUBLICATIONS IN REFEREED JOURNALS

RESEARCH ARTICLES

1. Sanchez A, Trappier SG, **Stroher U**, Nichol ST, Bowen MD, Feldmann H: Variation in the glycoprotein and VP35 genes of Marburg virus strains. *Virology* 1998, 240:138-146.
2. Volchkov VE, Volchkova VA, **Stroher U**, Becker S, Dolnik O, Cieplik M, Garten W, Klenk HD, Feldmann H: Proteolytic processing of Marburg virus glycoprotein. *Virology* 2000, 268:1-6.
3. **Stroher U**, West E, Bugany H, Klenk HD, Schnittler HJ, Feldmann H: Infection and activation of monocytes by Marburg and Ebola viruses. *J Virol* 2001, 75:11025-11033.
4. Groseth A, **Stroher U**, Theriault S, Feldmann H: Molecular characterization of an isolate from the 1989/90 epizootic of Ebola virus Reston among macaques imported into the United States. *Virus Res* 2002, 87:155-163.
5. Krokhin O, Li Y, Andonov A, Feldmann H, Flick R, Jones S, **Stroeher U**, Bastien N, Dasuri KV, Cheng K, et al: Mass spectrometric characterization of proteins from the SARS virus: a preliminary report. *Mol Cell Proteomics* 2003, 2:346-356.
6. Marra MA, Jones SJ, Astell CR, Holt RA, Brooks-Wilson A, Butterfield YS, Khattra J, Asano JK, Barber SA, Chan SY, Cloutier A, Coughlin SM, Freeman D, Girn N, Griffith OL, Leach SR, Mayo M, McDonald H, Montgomery SB, Pandoh PK, Petrescu AS, Robertson AG, Schein JE, Siddiqui A, Smailus DE, Stott JM, Yang GS, Plummer F, Andonov A, Artsob H, Bastien N, Bernard K, Booth TF, Bowness D, Czub M, Drebot M, Fernando L, Flick R, Garbutt M, Gray M, Grolla A, Jones S, Feldmann H, Meyers A, Kabani A, Li Y, Normand S, **Stroher U**, Tipples GA, Tyler S, Vogrig R, Ward D, Watson B, Brunham RC, Krajden M, Petric M, Skowronski DM, Upton C, Roper RL. The Genome sequence of the SARS-associated coronavirus. *Science* 2003, 300:1399-1404.
7. Takada A, Feldmann H, **Stroeher U**, Bray M, Watanabe S, Ito H, McGregor M, Kawaoka Y: Identification of protective epitopes on ebola virus glycoprotein at the single amino acid level by using recombinant vesicular stomatitis viruses. *J Virol* 2003, 77:1069-1074.
8. Dolnik O, Volchkova V, Garten W, Carbonnelle C, Becker S, Kahnt J, **Stroher U**, Klenk HD, Volchkov V: Ectodomain shedding of the glycoprotein GP of Ebola virus. *Embo J* 2004, 23:2175-2184.
9. Garbutt M, Liebscher R, Wahl-Jensen V, Jones S, Moller P, Wagner R, Volchkov V, Klenk HD, Feldmann H, **Stroher U**: Properties of replication-competent vesicular stomatitis virus vectors expressing glycoproteins of filoviruses and arenaviruses. *J Virol* 2004, 78:5458-5465.
10. He R, Leeson A, Ballantine M, Andonov A, Baker L, Dobie F, Li Y, Bastien N, Feldmann H, **Strocher U**, et al: Characterization of protein-protein interactions between the nucleocapsid protein and membrane protein of the SARS coronavirus. *Virus Res* 2004, 105:121-125.
11. **Stroher U**, DiCaro A, Li Y, Strong JE, Aoki F, Plummer F, Jones SM, Feldmann H: Severe acute respiratory syndrome-related coronavirus is inhibited by interferon- alpha. *J Infect Dis* 2004, 189:1164-1167.
12. Geisbert TW, Jones S, Fritz EA, Shurtleff AC, Geisbert JB, Liebscher R, Grolla A, **Stroher U**, Fernando L, Daddario KM, et al: Development of a new vaccine for the prevention of Lassa fever. *PLoS Med* 2005, 2:e183.
13. Jones SM, Feldmann H, **Stroher U**, Geisbert JB, Fernando L, Grolla A, Klenk HD, Sullivan NJ, Volchkov VE, Fritz EA, et al: Live attenuated recombinant vaccine protects nonhuman primates against Ebola and Marburg viruses. *Nat Med* 2005, 11:786-790.
14. Wahl-Jensen V, Kurz SK, Hazelton PR, Schnittler HJ, **Stroher U**, Burton DR, Feldmann H: Role of Ebola virus secreted glycoproteins and virus-like particles in activation of human macrophages. *J Virol* 2005, 79:2413-2419.
15. Wahl-Jensen VM, Afanasieva TA, Seebach J, **Stroher U**, Feldmann H, Schnittler HJ: Effects of Ebola virus glycoproteins on endothelial cell activation and barrier function. *J Virol* 2005, 79:10442-10450.

16. Daddario-DiCaprio KM, Geisbert TW, Geisbert JB, **Stroher U**, Hensley LE, Grolla A, Fritz EA, Feldmann F, Feldmann H, Jones SM: Cross-protection against Marburg virus strains by using a live, attenuated recombinant vaccine. *J Virol* 2006, 80:9659-9666.
17. Daddario-DiCaprio KM, Geisbert TW, **Stroher U**, Geisbert JB, Grolla A, Fritz EA, Fernando L, Kagan E, Jahrling PB, Hensley LE, et al: Postexposure protection against Marburg haemorrhagic fever with recombinant vesicular stomatitis virus vectors in non-human primates: an efficacy assessment. *Lancet* 2006, 367:1399-1404.
18. Hoenen T, Groseth A, Kolesnikova L, Theriault S, Ebihara H, Hartlieb B, Bamberg S, Feldmann H, **Stroher U**, Becker S: Infection of naive target cells with virus-like particles: implications for the function of ebola virus VP24. *J Virol* 2006, 80:7260-7264.
19. Towner JS, Khristova ML, Sealy TK, Vincent MJ, Erickson BR, Bawiec DA, Hartman AL, Comer JA, Zaki SR, **Stroher U**, et al: Marburgvirus genomics and association with a large hemorrhagic fever outbreak in Angola. *J Virol* 2006, 80:6497-6516.
20. Bockeler M, **Stroher U**, Seebach J, Afanasieva T, Suttorp N, Feldmann H, Schnittler HJ: Breakdown of paracndothelial barrier function during Marburg virus infection is associated with early tyrosine phosphorylation of platelet endothelial cell adhesion molecule-1. *J Infect Dis* 2007, 196 Suppl 2:S337-346.
21. Feldmann H, Jones SM, Daddario-Dicaprio KM, Geisbert JB, **Stroher U**, Grolla A, Bray M, Fritz EA, Fernando L, Feldmann F, et al: Effective Post-Exposure Treatment of Ebola Infection. *PLoS Pathog* 2007, 3:e2.
22. Groseth A, Hoenen T, Alimonti JB, Zielecki F, Ebihara H, Theriault S, **Stroher U**, Becker S, Feldmann H: In vitro evaluation of antisense RNA efficacy against filovirus infection, by use of reverse genetics. *J Infect Dis* 2007, 196 Suppl 2:S382-389.
23. Jones SM, **Stroher U**, Fernando L, Qiu X, Alimonti J, Melito P, Bray M, Klenk HD, Feldmann H: Assessment of a vesicular stomatitis virus-based vaccine by use of the mouse model of Ebola virus hemorrhagic fever. *J Infect Dis* 2007, 196 Suppl 2:S404-412.
24. **Stroher U**, Willihnganz L, Jean F, Feldmann H: Blockage of filoviral glycoprotein processing by use of a protein-based inhibitor. *J Infect Dis* 2007, 196 Suppl 2:S271-275.
25. Geisbert TW, Daddario-Dicaprio KM, Geisbert JB, Reed DS, Feldmann F, Grolla A, **Stroher U**, Fritz EA, Hensley LE, Jones SM, **Feldmann H**: Vesicular stomatitis virus-based vaccines protect nonhuman primates against aerosol challenge with Ebola and Marburg viruses. *Vaccine* 2008, 26:6894-6900.
26. Lee AM, Rojek JM, Gundersen A, **Stroher U**, Juteau JM, Vaillant A, Kunz S: Inhibition of cellular entry of lymphocytic choriomeningitis virus by amphipathic DNA polymers. *Virology* 2008, 372:107-117.
27. Maisa A, **Stroher U**, Klenk HD, Garten W, Strecker T: Inhibition of Lassa virus glycoprotein cleavage and multicycle replication by site 1 protease-adapted alpha(1)-antitrypsin variants. *PLoS Negl Trop Dis* 2009, 3:e446.
28. Qiu X, Fernando L, Alimonti JB, Melito PL, Feldmann F, Dick D, **Stroher U**, Feldmann H, Jones SM: Mucosal immunization of cynomolgus macaques with the VSVDeltaG/ZEBOVGP vaccine stimulates strong ebola GP-specific immune responses. *PLoS One* 2009, 4:e5547.
29. Ritchie G, Harvey DJ, Feldmann F, **Stroeher U**, Feldmann H, Royle L, Dwek RA, Rudd PM: Identification of N-linked carbohydrates from severe acute respiratory syndrome (SARS) spike glycoprotein. *Virology* 2010, 399:257-269.
30. Ritchie G, Harvey DJ, **Stroeher U**, Feldmann F, Feldmann H, Wahl-Jensen V, Royle L, Dwek RA, Rudd PM: Identification of N-glycans from Ebola virus glycoproteins by matrix-assisted laser desorption/ionisation time-of-flight and negative ion electrospray tandem mass spectrometry. *Rapid Commun Mass Spectrom* 2010, 24:571-585.
31. Schlie K, Maisa A, Lennartz F, **Stroher U**, Garten W, Strecker T: Characterization of Lassa virus glycoprotein oligomerization and influence of cholesterol on virus replication. *J Virol* 2010, 84:983-992.

BOOK CHAPTERS & REVIEWS

32. Feldmann H, Volchkov VE, **Stroher U**, Klenk HD: Marburg and Ebola Viruses. *Nova Acta Leopoldina* 2000, NF80:163-178.
33. Feldmann H, Volchkov VE, Volchkova VA, **Stroher U**, Klenk HD: Biosynthesis and role of filoviral glycoproteins. *J Gen Virol* 2001, 82:2839-2848.
34. Feldmann H, Wahl-Jensen V, Jones SM, **Stroher U**: Ebola virus ecology: a continuing mystery. *Trends Microbiol* 2004, 12:433-437.
35. Schnittler H, **Stroher U**, Afanasieva T, Feldmann H: The Role of Endothelial Cells in Filoviral Hemorrhagic Fever. In *Ebola and Marburg Viruses: Moelcular and Cellular Biology*. Edited by Klenk HD, Feldmann H. Wymondham, U.K.: Horizon Bioscience; 2004
36. Wahl-Jensen V, **Stroher U**, Schnittler H, Feldmann H: Current Opinion on Ebola Virus Pathogenesis. *Nova Acta Leopoldina* 2005, 92:147-154.
37. **Stroher U**, Feldmann H: Progress towards the treatment of Ebola haemorrhagic fever. *Expert Opin Investig Drugs* 2006, 15:1523-1535.
38. Afanasieva T, Wahl-Jensen V, Seebach J, Schillers H, Nikova D, **Stroeher U**, Feldmann H, Schnittler H: Endothelial Cells and Ebola-virus hemorrhagic fever. In. New York: Cambridge University Press; 2007

NON-REFEREED PUBLICATIONS

39. Schnittler HJ, Gotsch U, Vestweber D, **Ströher U**, Feldmann H: Die Endothelzellausrichtung unter Schubspannung ist mit einer Reorganisation und Aktivierung von Komponenten der Zell-Zell-Kontakte assoziiert. *Ann Anat / Verh Anat Ges* 1998, 93.
40. **Ströher U**, Feldmann H, Schnittler H: Bedeutung von Monozyten/ Makrophagen und Endothelzellen für die Pathogenese schwerer hämorrhagischer Fieber bedingt durch Filoviren. *Ann Anat / Verh Anat Ges* 1998, 93.
41. **Ströher U**: Filovirus induced target cell activation. *Infect Dis Rev* 2001, 3.
42. Cordova JA, Hernandez M, Lopez-Gatell H, Hernandez JE, Tustin J, Watkins K, Stuart TL, Kuschak T, **Ströher U**, Soule G, et al: CDC Update: novel influenza A (H1N1) virus infection - Mexico, March-May, 2009. *MMWR Morb Mortal Wkly Rep* 2009, 58:585-589.

CONFERENCE PRESENTATIONS *(1-25: poster presentations; 26-70: oral presentations)*

1. **Ströher U.**, Sanchez A., Klenk H.D., Feldmann H. (1995). The Marburg group of filoviruses: genetic variability and characterization of a second ORF encoding a potential nonstructural protein. First European Meeting of Virology, Würzburg, Germany.
2. Volchkova V.A., Volchkov V.E., **Ströher U.**, Klenk H.D., LeGuenno B., Sanchez A., Feldmann H. (1997). Ebola virus outbreak in Gabon: Genetic variation is not the major factor in filovirus emergence. Jahrestagung der Gesellschaft für Virologie, Hamburg, Germany.
3. **Ströher U.**, Feldmann H., Schnittler H. (1997). Bedeutung von Monozyten/Makrophagen und Endothelzellen für die Pathogenese schwerer hämorrhagischer Fieber bedingt durch Filoviren. 14. Arbeitstagung der Anatomischen Gesellschaft, Würzburg, Germany.
4. **Ströher U.**, Feldmann H., Suttorp N., Schnittler H. (1998). Filovirus-induced endothelial cell activation. Symposium ‚Biology of the vessel wall', Münster, Germany.
5. Jensen V., **Ströher U.**, Feldmann H. (2001). Expression of Filovirus Glycoproteins. Faculty of Medicine at the University of Manitoba: Research Day.

6. Willihnganz L.J., Jean F., Feldmann H., **Ströher U.** (2003). Blockage of Filoviral Glycoprotein Processing using a Protein-based Inhibitor. NSV meeting, Pisa, Italy.
7. Willihnganz L.J., Jean F., Feldmann H., **Ströher U.** (2003). Blockage of Filoviral Glycoprotein Processing using a Protein-based Inhibitor. International Conference on Emerging Zoonoses. Ames, Iowa, USA.
8. Afanasieva T., Jensen V., Seebach J., **Ströher U.**, Feldmann H., Schnittler H.J. (2004). German Society for Biochemistry and Molecular Biology. Münster, Germany.
9. Vaillant A., **Ströher U.**, Evans D., Jones S., Feldmann H., Juteau J.M. (2005). Sequence-independent antiviral activity of Phosphorothioate Oligonucleotides against biodefense related viruses. The First Annual Meeting of the Oligonucleotide Therapeutics Society, NY, USA.
10. Camus G., **Ströher U.**, Jones S.M. (2006). Generation of Lassa virus-like particles for the development of an ELISA assay. 1st Annual Public Health Agency of Canada Research Forum, Winnipeg, MB, Canada.
11. Hoenen T., Groseth A., Hartlieb B., Feldmann H., **Ströher U.**, Becker B. (2006). Role of Ebola Virus VP24 and VP30 in ribonucleoprotein complex formation. 1st Annual Public Health Agency of Canada Research Forum, Winnipeg, MB, Canada.
12. Groseth A., Hoenen T., Becker B., **Ströher U.**, Feldmann H.(2006). Regulation of Filovirus Transcription by VP30: More than just a hairpin ? NSV meeting, Salamanca, Spain.
13. Camus G., **Ströher U.**, Strecker T., Hazelton P., Qiu X., Jones S. (2007). Generation and Characterization of Lassa Virus-Like Particles. Public Health Agency of Canada Research Forum, Winnipeg, MB, Canada.Camus G., **Ströher U.**, Strecker T., Hazelton P., Qiu X., Jones S. (2007). Generation of virus-like particles as a novel platform for vaccine development against Lassa fever. 13th International Congress of Immunology, Rio de Janeiro, Brazil.
15. Chan M., Balcewich B., Strecker T., Camus G., Studera X., Jones S.M., Feldmann H., **Ströher U.** (2008). Glycosylation of Machupo and Junin virus glycoprotein-2 is crucial for proteolytical processing of GPC and incorporation into virus-like particles. XIVth International Congress of Virology, Istanbul, Turkey
16. Schlie K., Freiberg F., **Stroeher U.**, Strecker T., Garten W. (2008). Directed release of Lassa virus-like particles in polarizd epithelial cells. XIVth International Congress of Virology, Istanbul, Turkey.
17. Maisa A., **Stroeher U.**, Garten W., Strecker T. (2008). Inhibition of Lassa virus glycoprotein maturation by highly specific antitrypsin variants reduces efficient virus replication. XIVth International Congress of Virology, Istanbul, Turkey.
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20. Judie Alimonti, **et al.** (2009) Live Replicating Viruses As Vaccines & Therapies For Viral Haemorrhagic Fever Viruses. 4th Public Health Agency of Canada Research Forum, Winnipeg, Canada.
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69. **Ute Ströher**, Judie Alimonti, Xiangguo Qiu, Lisa Fernando, Heinz Feldmann, Peter Jahrling, Lisa Hensley, Steven M Jones (2009). Development of Live Replicating Viruses as Vaccines and Therapies for Viral Haemorrhagic Fever Viruses. Medical Biodefense Conference, Munich, Germany
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INVITED SEMINARS (2008/09):

Ströher U. (2008). 'Progress' towards the treatment of Ebola haemorrhagic fever. Fundaciòn Ciencia Para La Vida, Santiago, Chile.

Ströher U. (2008). Antiviral Approaches against Viral Haemorrhagic Fever Viruses. Robert-Koch Institut, Berlin, Germany.

Ströher U. (2009). Viral Haemorrhagic Fever Viruses. CNRS and University Aix-Marseille I and II, Marseille, France

AWARDS:

2003: Health Canada Special Team Excellence Award: 2003 SARS Outbreak

2006: Public Health Agency of Canada Research Merit Award (Ebola/Marburg Hemorrhagic Fevers Vaccine)

INNOVATIONS

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FUNDING (current)

2006-2011:
Development of live replicating viruses as vaccines and therapies for Viral Hemorrhagic fever viruses.
CRTI [Co-Investigator/Project Manager]

2008-2013:
Development of Canadian diagnostic capability for Rift Valley fever virus (RVFV).
CRTI [Co-Investigator]

2009-2010:
Rapid Sequencing capability in the field for confirmation of identification.
CRTI [Principal Investigator]